Cardiac Function and Mean Circulatory Pressure in Acute Impediment to Venous Return

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THE classical backward failure theory maintains that the clinical manifestations of congestive heart failure are due to passive engorgement of the venous tributaries, caused by "backward" rise in pressure, retrograde from the failing cardiac chamber into the venous system. That this "backward rise in pressure" is mainly due to an increased volume of blood in association with an increase of venous tone has been shown for local venous beds in human congestive heart failure (WOOD 1956, BURCH 1954).

It will be worthwhile, if it can be shown in acute animal experiments that the blood is never simply dammed up behind the right atrium, when the venous return is acutely impeded by a balloon in the right atrium. A useful method serving for this purpose is the measurement of the mean circulatory pressure (MCP), a quantity and a concept thoroughly studied by GUYTON and his colleagues (GUYTON 1955, 1959, 1963a, 1963b). The mean circulatory pressure (MCP) is a measure of the ratio of the blood volume to the capacity of the circulatory system and it represents the degree of filling of the circulatory system with blood (GUYTON et al., 1956). Thus the increase in MCP in the absence of any change in blood volume must be due to a decrease in the capacity of the circulatory system. If the elevated venous pressure in acute right-sided heart failure is due to a passive translocation of the blood from the arterial circuit into the venous circuit, there will be no change in the MCP. An increased MCP means an increased vis-a-tergo for venous return, since the MCP was shown to represent the mean driving pressure returning blood to the heart (GUYTON 1955).

Hemodynamic responses of open-chest dogs to progressive central venous resistance were studied by GUYTON (1952). The present study is an attempt to evaluate, in open-chest dogs, the changes in MCP, superior vena cava and peripheral (leg) venous pressures and other circulatory parameters that occur when the venous return is acutely impeded by a balloon in the right atrium, with emphasis on the numerical relationship between the peripheral venous pressure and the MCP.

METHODS

The method used to measure the MCP was similar to that described by SMITH (1967), but the present experiments were performed on open-chest dogs.

Eight mongrel dogs weighing between 12 and 18 kg were used. The dogs were anesthetized with 30 mg/kg sodium pentobarbital and heparinized with 2.0–2.5 mg/kg of sodium heparin. The chest was opened at the 4th left intercostal space under artificial respiration. The pericardium was opened with electrocautery and an electromagnetic flow probe, model MF-5, Nihon Kohden Kogyo Co. was placed around the root of the aorta to measure the cardiac output (excluding coronary flow). A pair of hook electrodes was placed on the right or left atrial appendage about 8 mm apart, so that the heart could be driven electrically when desired, e.g., for a short period of time immediately after the heart was defibrillated. The artificial respiration was continued throughout this experiment. The electrocardiogram was also recorded.

The right jugular vein and bilateral femoral

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arteries and veins were exposed and cannulated with polyethylene tubing. Two catheters were introduced through the right jugular vein. One catheter provided with an inflatable balloon in the tip was advanced, under fluoroscopic monitoring, until the tip was located in the right atrium. When inflated, the contour of the balloon could be readily made out fluoroscopically. Requirements for a satisfactory balloon inflation was (1) position of the balloon in the right atrium and (2) reasonable matching of the superior vena cava pressure with the foreleg and iliac venous pressure. Another catheter was advanced, under fluoroscopic monitoring, until the pressure tracing as well as the fluoroscopic findings were compatible with the location of the catheter tip within the superior vena cava.

The two catheters in the femoral arteries were advanced until their tips reached the descending aorta beyond the level of the diaphragm. These two femoral arterial catheters were connected to the input side of a cum-pump, model C-16, Tokyo Rikakikai Co.. The output of the pump was connected with the catheters in femoral veins, advanced until their tips were within the common iliac veins. When activated, the pump could pump blood rapidly from the arterial circuit into the venous circuit at a rate of 40 ml/sec.

A superficial vein of the unrestrained foreleg was punctured and this system was connected, via a three-way stop-cock, with a saline manometer of a reasonably short response time (10 sec). The remaining arm of the three-way stop-cock was connected with a side arm of the femoral vein catheter, so that the pressure in the common iliac vein could be measured in succession with the foreleg venous pressure measurements.

The right carotid artery was cannulated with a T-tube to measure the lateral pressure of this artery.

Superior vena cava pressure and carotid artery pressure were recorded on a multipurpose polygraph, model RM-150, Nihon Kohden Kogyo, Co., using pressure transducers, MP-24T, Nihon Kohden Kogyo, Co. The zero reference point for the pressure measurements was set, under direct visual control, at the level of the right atrium.

The heart was fibrillated with a shock of 2 volts AC applied directly to the ventricular walls. Immediately after inducing fibrillation, blood was translocated from the femoral arteries to the femoral veins by activating the previously described pump. When blood was translocated in this manner, the systemic arterial pressure fell

and vena cava pressure rose. The point at which the two pressures equal was termed the mean circulatory pressure (MCP). In most cases, the MCP was measured within 4 sec of the induction of ventricular fibrillation. Once the pressure reached equilibrium, the heart was defibrillated with a shock of 30 watt sec DC applied through a pair of plate-electrodes in direct contact with the heart. When the heart was then found in standstill, it was driven with square pulses of 5 volts and 5 msec duration at frequency of 1.0–1.5 cycle per sec, until it began beating spontaneously. The animal was then allowed sufficient recovery time for the measured parameters to return to their control values.

Control measurements were obtained in each of the following parameters: cardiac output, arterial pressure, superior vena cava pressure, foreleg and iliac venous pressure and the MCP. The right atrial balloon was inflated and the result was a marked fall in arterial pressure and cardiac output, concomitant with a large increase in the superior vena cava pressure and a proportional increase in the foreleg and iliac venous pressure. At the end of 3–5 minutes after the start of the systemic "venous congestion", MCP and the various other measurements were made and the heart was defibrillated, the balloon deflated. The term "venous congestion" will be used in this paper as a synonym of a "rise in venous pressure".

It was ascertained with dye-dilution studies that the measurement of the cardiac output with electromagnetic flow probes of non-cannulating type was generally valid, although with somewhat reduced accuracy, at mean arterial blood pressures as low as 10–60 mmHg (Hirakawa, S. et al, manuscript in preparation).

In 4 of the 8 dogs, the MCP and other parameters were measured before and during acute systemic "venous congestion", and the results were repeated again after their autonomic reflexes have been abolished markedly, if not completely, by the intravenous injection of pentolinium tartrate (Ansolsen) in dose of 0.35 mg/kg. Control measurements after pentolinium were obtained without rectifying the pentolinium-induced fall of arterial pressure, cardiac output and the MCP, and they were then subjected to acute systemic "venous congestion".

The extent of the blood volume change during the "venous congestion" was measured in 3 dogs other than these 8 dogs. In these dogs measurements were made of hematocrit, MCP and other circulatory parameters before and during the

"venous congestion". After the animals were allowed sufficient recovery time, measurements were again made of hematocrit, MCP and other circulatory parameters before and during a severer "venous congestion".

Abbreviations and symbols to be used in this paper are as follows: CO = Cardiac output (ml/min); CVP = Central venous pressure (mmHg), as measured in the superior vena cava; GVR = Gradient for venous return (mmHg). By definition in this paper, $GVR = MCP - CVP$; MCP = Mean circulatory pressure, in mmHg, as measured in open-chest dogs by stopping the heart and rapidly translocating blood from the arterial into venous circuit until $Pa = CVP$; Pa = Mean arterial pressure (mmHg); RVR = Resistance for venous return ($\text{dyne} \cdot \text{sec} \cdot \text{cm}^{-5}$). It must be noted that the resistance to flow offered by the balloon in the right atrium is not included in RVR; TPR = Total pe-

ripheral resistance ($\text{dyne} \cdot \text{sec} \cdot \text{cm}^{-5}$), calculated as $Pa - CVP$ divided by CO; VP_f = Foreleg venous pressure (mmHg); VP_i = Iliac venous pressure (mmHg).

RESULTS

Reproducibility of the MCP and other circulatory parameters during the control period

In the present series of experiments in open-chest dogs, the mean arterial pressure was usually 80 ± 10 mmHg, but no attempt was made to keep the mean arterial pressure above 100 mmHg with the infusion of pressor substances. Under this circumstance, the MCP ranged between 5 and 6 mmHg and CO about 1 L/min. A preliminary study on the reproducibility of MCP measurements in one and the same such open-chest dog showed that the MCP measurements repeated 7 times during the course of 35 minutes gave a

Fig. 1. Typical experiment showing the change in Pa, CVP, CO and the measurement of MCP during the systemic "venous congestion" produced by inflating a balloon in the right atrium. Tracings are, from above downwards: ECG, differential manometer output, aortic flow, arterial pressure, superior vena cava pressure, integrated aortic flow (each step representing stroke volume). The gain of the electromanometer monitoring the arterial pressure was changed, as indicated in the figure, in order to bring the differential manometer into action. Zero differential manometer output means equal pressures in the carotid artery and the superior vena cava and this pressure is the MCP.

Time calibration, 1 sec. It is seen that the time interval from the start of ventricular fibrillation to the equalization of arterial and caval venous pressure was 2.8 sec and MCP 8 mmHg.

TABLE I MEAN CIRCULATORY PRESSURE, CARDIAC OUTPUT AND OTHER CIRCULATORY PARAMETERS IN CONTROL PERIOD AND DURING ACUTE SYSTEMIC "VENOUS CONGESTION" PRODUCED BY INFLATING A BALLOON IN THE RIGHT ATRIUM

	Control	"Venous Congestion"	
		Mild	Severe
No. of measurements	8	10	5
Pa mmHg	86 ± 5.5	53 ± 5.8	38 ± 9.7
CVP mmHg	0.2 ± 0.1	4.2 ± 0.5	8.8 ± 1.3
CO ml.min ⁻¹	100% (990 ± 160)	55 ± 7.1	24 ± 6.2
MCP mmHg	5.4 ± 0.4	9.0 ± 1.0	12.4 ± 0.9
TPR dyne.sec.cm ⁻⁵	100% (8500 ± 1450)	114 ± 18.2*	219 ± 108*
GVR mmHg	5.2 ± 0.4	4.9 ± 0.9*	3.6 ± 0.8*
RVR dyne.sec.cm ⁻⁵	100% (550 ± 150)	266 ± 85.1*	316 ± 58.0
VP _f mmHg	1.4 ± 0.4	4.4 ± 0.4	10.1 ± 1.0
VP _i mmHg	1.0 ± 0.1	4.7 ± 0.4	10.4 ± 1.0

For abbreviations, see "Methods." Measurements made during "venous congestion" are given in two groups, separated arbitrarily according as the foreleg VP exceeded 7.4 mmHg or not. Asterisk signs indicate that the difference from the control is not statistically significant, $P > 0.05$. Variance is expressed in standard error of the mean.

mean value of 5.4 mmHg with 0.4 mmHg standard deviations. Details of this study will be described in a separate paper (HIRAKAWA, S. et al., manuscript in preparation) together with the influence of pentobarbital and phenylephrine on the mean circulatory pressure in open-chest dogs.

Changes in the MCP and other circulatory parameters during acute systemic "venous congestion"

Before ganglionic blockade:

Fig. 1 shows a typical experiment.

The results obtained in these experiments on 8 open-chest dogs are shown in Table I. The measurements obtained during the acute "venous congestion" are arbitrarily separated into 2 groups according as the foreleg venous pressure was equal to, or exceeded, 7.4 mmHg (10 cm H₂O), termed the severe "venous congestion", or it did not reach 7.4 mmHg (10 cm H₂O), termed the mild "venous congestion".

It is estimated, from hematocrit studies in another series of 3 dogs, that there was an average of $6 \pm 3\%$ (SD) estimated decrease in the blood volume in 4–5 minutes of severe "venous congestion". In this instance the average rise of the foreleg venous pressure was 10 mmHg above the control level.

There was no systematic difference between the arterial blood hematocrit change and the venous blood hematocrit change. By contrast, with mild "venous congestion" of 4–5 minute duration, there was no appreciable change in hematocrit except in few instances.

Since there was no increase, but a slight decrease, in the estimated blood volume during the "venous congestion", it is concluded from this study that the acute "venous congestion" produced by inflating a balloon in the right atrium was accompanied by a marked decrease in the capacitance of the circulatory system. The responsible mechanisms may be (1) vasoconstriction and (2) where hemoconcentration occurred, an elevated tissue pressure resulting from augmented fluid filtration.

After ganglionic blockade:

The balloon experiment was repeated, in 4 of the 8 dogs, after their autonomic reflexes were abolished markedly, if not completely, with pentolinium tartrate (Ansolsen). The results in the ganglioplegic dogs are shown in Table II. Inspection of this table in comparison with Table I indicates that the ganglionic blockade, per se, caused the arterial pressure, total peripheral resistance and the MCP to fall considerably and the cardiac output to decrease slightly. When the animals were then subjected to the balloon inflation, the result was usually a mild "venous congestion", mild in that the foreleg venous pressure did not reach 7.4 mmHg (10 cm H₂O). Table II summarizes 4 such results. The mild "venous congestion" obtained in the ganglioplegic dogs simulated that obtained in normal dogs in parts. Thus nearly the same cardiac output, 43 ± 8.9 versus $55 \pm 7.1\%$ of the pre-pentolinium control value, the same CVP, 3.9 ± 0.1 versus 4.2 ± 0.5 mmHg, and the same peripheral venous pressures

TABLE II MEASUREMENTS IN DOGS WITH GANGLIONIC BLOCKADE BEFORE AND DURING THE SYSTEMIC "VENOUS CONGESTION" PRODUCED BY INFLATING A BALLOON IN THE RIGHT ATRIUM

	Control	Mild "Venous Congestion"
No. of measurements	4	4
Pa mmHg	57 ± 5	25 ± 5.2
CVP mmHg	0.8 ± 0.3	3.9 ± 0.1
CO ml.min ⁻¹	79.9 ± 13.5% (870 ± 220)	43 ± 8.9
MCP mmHg	4.3 ± 0.5	5.9 ± 0.5
TPR dyne·sec·cm ⁻⁵	100% (3770 ± 1010)	79 ± 20.3*
GVR mmHg	3.5 ± 0.8	2.0 ± 0.6*
RVR dyne·sec·cm ⁻⁵	100% (440 ± 180)	111 ± 30.6*
VP _f mmHg	2.1 ± 0.8	4.4 ± 0.3
VP _i mmHg	1.1 ± 0.5	4.3 ± 0.3

For abbreviations, see "Methods". The "venous congestion" data with the foreleg VP less than 7.4 mmHg are shown. Cardiac output (CO) is expressed in percent of the control measurements made prior to the ganglionic blockade. Asterisk signs indicate that the difference from the control is not statistically significant. Variance is expressed in standard error of the mean.

Fig. 2. Alterations in the "cardiac output curve" and "venous return curve" constructed from data obtained in these experiments. Horizontal and vertical bars indicate standard errors of the mean. Dark solid line represents the "venous return curve" in the control period. The long-dashed "venous return curve" was obtained after inflating a balloon in the right atrium when the resulting "venous congestion" was mild in that the foreleg venous pressure did not reach 7.4 mmHg (10 cm H₂O). The short-dashed "venous return curve" was obtained when "venous congestion" was severe in that the foreleg venous pressure exceeded 7.4 mmHg (10 cm H₂O). The point at which "venous return curve" intersects the abscissa represents the MCP.

were recorded during the mild "venous congestion" with or without ganglionic blockade. In spite of these similarities, there was a considerable difference in the magnitude of the increase in the MCP during the "venous congestion". Thus the increase in MCP was from 4.3 to 5.9 mmHg ($0.02 < P < 0.05$) in ganglioplegic dogs, while it increased from 5.4 to 9.0 mmHg ($0.001 < P <$

0.005) in normal dogs.

DISCUSSION

It was demonstrated by the present experiment that the "venous congestion" produced by inflating a balloon in the right atrium of open-chest dogs was associated with a distinct decrease in the capacitance of the circulatory system. This

Fig.3. "Cardiac output curve" and "venous return curve" before and during a mild "venous congestion", (a) before ganglionic blockade and (b) after ganglionic blockade. Dark solid lines represent the "venous return curves" in the control periods. The long-dashed "venous return curves" were obtained during "venous congestion" of mild degree where the foreleg venous pressure did not reach 7.4 mmHg (10 cm H₂O). Cardiac output is expressed in percent of the "initial" control value obtained before ganglionic blockade. Note that the MCP rose during the "venous congestion" in ganglioplegic dogs, but to a lesser extent than in normal dogs.

Fig.4. Circulatory changes during the acute systemic "venous congestion" illustrated with a model of the circulatory system. Arrows oriented upwards and downwards indicate an increase and decrease, respectively. A shift downward to the right of the "cardiac output curve" is expressed as a decrease in the output function (OF). When the venous return was impeded, there occurred a decrease in the OF, associated with an increase in MCP, with other changes as shown in this figure.

Fig.5. A schematic illustration of the alterations in the circulatory system during the mild "venous congestion" with or without ganglionic blockade. With ganglionic blockade, a decrease in the output function of the heart (OF) was no longer associated with a marked increase in MCP.

conclusion is inevitable from the fact that there was a clear increase in the MCP in the absence of any increase in the blood volume. This experiment also disclosed the manner in which the cardiac output is regulated by the organism in face of a balloon impeding the systemic venous return to the heart. Fig. 2 shows the alterations in "cardiac output curve" and "venous return curve" during the acute "venous congestion". In Fig. 2 and 3 the cardiac output curve and venous return curve were modified to correlate the cardiac output and venous return, respectively, with the superior vena cava pressure, instead of right atrial pressure. This modification will be justifiable as long as the pressure just peripheral to the balloon is regarded as the filling pressure for the "heart" and, at the same time, the vis-a-fronte for the venous return. In face of a marked shift downwards to the right of the "cardiac output curve", such as seen in Fig. 2 during the "venous congestion", a drastic decrease in cardiac output would have been inevitable, if it were not for a shift to the right of the "venous return curve". Thus, the shift to the right of the "venous return curve" that occurs during "venous congestion" and shown in Fig. 2 and 3, obviously serves a compensatory purpose to bring back the cardiac output as much towards normal as possible.

Fig. 3 shows the extent to which the decrease in the capacitance of the circulatory system during acute "venous congestion" is abolished by the ganglionic blockade. Comparison of Fig. 3-a with Fig. 3-b would indicate that the vasoconstriction mediated through autonomic nervous system was the major factor contributing to the decrease in the capacitance (increase in the MCP) of the circulatory system during acute "venous congestion" in normal dogs. A small increase in the MCP during the "venous congestion" seen in ganglioplegic dogs may be due to residual activity of the nervous system, but other possibilities, such as the effect of a blood shift from the lung affecting the measured value of MCP, must be considered.

From Fig. 2 and 3 it is apparent that there are at least two mechanism by which the central venous pressure is elevated in acute impediment to the venous return in the absence of appreciable changes in blood volume, (1) impaired "cardiac function" and (2) increased vasomotor tone. The first, or "cardiac" factor seems to predominate during the mild "venous congestion", and this explains the reason why ganglionic blockade failed to influence the elevation of the central

venous pressure during mild "venous congestion".

Fig. 4 and 5 illustrate the data obtained in these experiments schematically, using a model of the circulatory system based on Starling's heart-lung preparation and incorporating the concept of the mean circulatory pressure (MCP). This oversimplified model must be viewed with caution, because this model is intended for simplicity at the cost of factual accuracy. For instance, the resistance for venous return (RVR) and the "total peripheral resistance" ("TPR") were incorporated, as if they were independent variables, while, in fact, they are not, the RVR taking care of both the arterial and the venous resistance to flow.

When the venous return was acutely impeded by inflating a balloon in the right atrium, the output function of the heart (OF) was depressed in that the "right ventricular output curve" shifted downward to the right. This change in "cardiac function" was associated with a marked increase in the MCP in normal dogs, as schematically shown in Fig. 4, but this response was no longer present in ganglioplegic dogs as shown in Fig. 5. The "TPR" during mild "venous congestion" was lower in ganglioplegic dogs (4560 ± 1000 dynes-sec-cm⁻⁵) than in normal dogs (8960 ± 1130 dynes-sec-cm⁻⁵).

Fig. 6 shows the numerical relationship between the MCP and the foreleg venous pressure obtained before and during the "venous congestion

Fig. 7. Results after ganglionic blockade, showing the numerical relationship between the MCP and foreleg venous pressure before and during the "venous congestion" produced by inflating a balloon in the right atrium.

tion" produced by an inflated balloon in the right atrium. There was a roughly parallel increase in MCP and peripheral venous pressure during the "venous congestion" of mild degree, but the MCP and peripheral venous pressure tended to more or less converge during severe "venous congestion". The over-all relation is expressed by a regression line, $MCP = 5 + 0.8 VP$.

Fig. 7 shows the results obtained with ganglionic blockade by pentolinium tartrate. The data for "venous congestion" were obtained from 4 measurements with mild "venous congestion" and 2 measurements during severe "venous congestion". The peripheral venous pressure was slightly closer to the MCP over the entire studied range of peripheral venous pressure than in normal dogs, and the peripheral venous pressure and MCP became very close to each other when the "venous congestion" was such that the foreleg venous pressure exceeded 7.4 mmHg (10 cm H₂O). This tendency is reflected by a regression line, $MCP = 3 + 0.7 VP$.

It is an interesting coincidence that the present author found, in man, a similar numerical relation between the cubital venous pressure (VP_c) and the mean systemic filling pressure estimated graphically under a few simplifying assumptions, termed Pms.gr. Thus, in 44 human subjects including normal subjects and patients with heart failure, the regression line was $Pms.gr. = 3 + 0.8 VP_c$ (HIRAKAWA, S., 1968a, 1968b).

Table III indicates that the diminished differ-

TABLE III ANALYSIS OF THE PRESSURE DIFFERENCE, $MCP - VP_f$, DURING MILD "VENOUS CONGESTION"

	$RVR \leq 440$ (dyne.sec.cm ⁻⁵)	$RVR > 440$ (dyne.sec.cm ⁻⁵)
$MCP - VP \leq 2$ mmHg	6	0
$MCP - VP > 2$ mmHg	0	8

All data in normal and ganglioplegic dogs were analyzed. The relation between the diminished pressure difference and low resistance for venous return (RVR) is statistically significant ($P < 0.005$).

ence between the MCP and peripheral venous pressure, $MCP - VP_f \leq 2$ mmHg, is associated with low resistance for venous return (RVR). This means that a relatively low RVR, associated with a substantial decrease in cardiac output (CO), explains those measurements made during mild "venous congestion", with or without ganglionic blockade, where the foreleg venous pressure was close to the numerical value of MCP. On the other hand, a severely diminished cardiac output is responsible for the fact that the foreleg venous pressure approaches the numerical value of MCP during severe "venous congestion" with or without ganglionic blockade.

The gradient for venous return (GVR) was shown to be proportional to the difference between mean circulatory pressure (MCP) and right atrial pressure (RAP), $GVR \approx MCP - RAP$ (GUYTON 1955). In this paper, gradient for venous return (GVR) is, by definition, MCP minus central venous pressure (CVP), thus $GVR = MCP - CVP$.

In open-chest dogs, the peripheral venous pressure was very close to the central venous pressure, the difference being but 1 mmHg on the average. Therefore the difference between the MCP and peripheral venous pressure is close to GVR.

Since the gradient for venous return (GVR) is a product of the venous return (VR) and resistance for venous return (GUYTON 1955) $GVR = VR \times RVR$, and since venous return is equal to cardiac output (CO) in steady-state conditions, such as prevailing in these experiments, $GVR = CO \times RVR$. Thus the result shown in Table III as well as the diminished pressure difference, $MCP - VP_f$, during severe "venous congestion", are in accord with theory. But this experiment must be extended to cover a greater number of animals before a definite statement can be made as to the quantitative relation between the MCP and pe-

peripheral venous pressure during the impediment to venous return.

If, in man, the chronic, right ventricular failure is not associated with a marked increase in the (systemic) resistance for venous return (RVR) as compared with normal RVR, the (systemic) gradient for venous return (GVR), and therefore the difference between the MCP and peripheral venous pressure, will tend to diminish with decreasing cardiac output. Under this circumstance, the peripheral venous pressure will be numerically closer to the MCP in the right heart failure than in normal heart. Exactly this relation was found by the present author in 44 human subjects with normal or abnormal heart, when the cubital venous pressure was compared with the mean systemic filling pressure estimated graphically under a few simplifying assumptions (HIRAKAWA, S. 1968a, 1968b). However, whether the chronic, right ventricular failure in man is not associated with a marked increase in the (systemic) resistance for venous return, remains to be decided.

SUMMARY

In order to evaluate the response of the circulatory system that occurs when the venous return to the right heart is acutely impeded by a balloon in the right atrium, measurements were made of the mean circulatory pressure (MCP), cardiac output and other circulatory parameters in open-chest dogs before and during acute artificial "venous congestion" produced by the right atrial balloon. The results were as follows:

(1) When the venous return was acutely impeded, there occurred a marked increase in the MCP, while blood volume decreased slightly or remained unchanged. This means that the capacity of the circulatory system has decreased.

(2) This response was abolished markedly, but not completely, when the animals were pre-treated with a ganglion-blocking agent, pentolinium tartrate (Ansolysen), although the "venous congestion" after ganglionic blockade was comparable with that obtained before ganglionic blockade with respect to cardiac output and central and peripheral venous pressures.

(3) This study showed that there are at least two mechanisms by which the venous pressure is elevated in acute impediment to the venous return, (a) impaired "cardiac function" and (b) increased vasomotor tone.

(4) When the venous return was impeded, the peripheral venous pressure rose nearly in parallelism with the MCP, but the venous pressure tended to approach the numerical value of MCP when the impediment to venous return was severe. Possible mechanisms responsible for this tendency were discussed.

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